

CASE REPORT

Primary human parvovirus B19 infection in an HIV infected patient on highly active antiretroviral therapy

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Adults developing primary human parvovirus B19 (B19) infection may present with arthralgia, fever, and maculopapular rash. Recovery is linked to the development of specific neutralising antibodies. In immunosuppressed patients, including those with HIV infection, such humoral responses are impaired and severe chronic bone marrow suppression and arthritis may occur.

Treatment of HIV by highly active antiretroviral therapy (HAART) has been shown to stimulate reconstitution of humoral and cell mediated immune competence¹ to various opportunistic infections.

A 36 year old male patient on HAART had a clinical course of primary B19 infection more in keeping with infection in immunocompetent adults than in those with untreated HIV infection, with the documented appearance of serum parvovirus DNA and subsequent antibody formation in serial monitoring over 12 months after the primary infection.

The patient was diagnosed as HIV positive in 1992 and had remained well; he was intolerant of attempted antiretroviral therapy which was offered several times between 1995 and 1997. In January 2001, he decided to restart therapy; his viral load was 100 000 copies/ml and peripheral CD4 lymphocyte count was $230 \times 10^6/l$. By week 16 of treatment with zidovudine, lamivudine, and efavirenz his viral load became undetectable on ultrasensitive testing but the CD4 count had dropped to $110 \times 10^6/l$.

Three months into the new HAART regimen, he developed a maculopapular rash on the arms and trunk, with fever, muscle and joint pains, anorexia, nausea, and cough.

His CD4 count was $180 \times 10^6/l$, and his viral load was 977 copies/ml. Human parvovirus B19 IgM and IgG detection was positive, consistent with recent infection with B19. Parvovirus B19 dot-blot and PCR DNA were positive on acute samples and negative on serum samples from 4 weeks and 12 weeks before presentation, supporting the diagnosis of primary B19 infection. Screening for other viral infections and syphilis was negative.

A transient drop in haemoglobin was noted in serial estimations (fig 1) but symptomatic anaemia did not develop. He was offered oral antipyretics and analgesics, and was advised to rest. Clinical recovery was complete within 4 weeks. Eighteen weeks after onset he was B19 IgM negative and IgG positive. Eight months after his illness the B19 DNA dot-blot and PCR were positive once more, with specific IgG and IgM detected. Twelve months after the rash, he remained well, but had discontinued HAART; CD4 count was $98 \times 10^6/l$ and viral load was 56 400 cpm.

Up to 25% of severe chronic anaemia in AIDS has been ascribed to B19 infection.^{2,3} The introduction of HAART is associated with reconstitution of immune responses, including humoral immunity. Detection of B19 infection in HIV infected patients depends on molecular amplification tech-

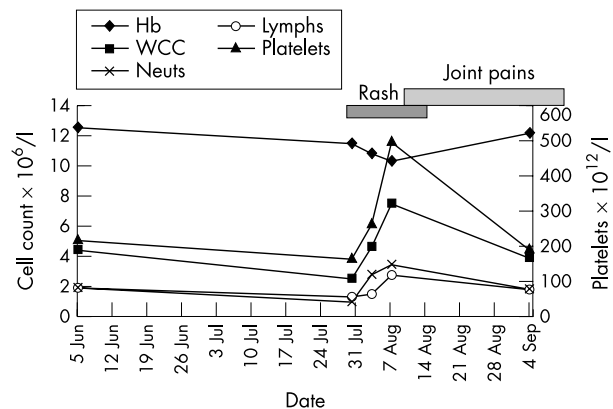


Figure 1 Course of haematological changes and symptoms.

niques rather than antibody responses.⁴ Initiating HAART has been beneficial in reversing B19 associated anaemia,⁵ and is linked with the return of humoral responses. In this case, a primary B19 infection was diagnosed in an HIV seropositive patient already on HAART. Humoral and viral markers indicated that immune responses to B19 developed, bone marrow suppression was mild and transient, and no chronic sequelae had been noted at up to 12 months of follow up.

Exposure to community outbreaks of B19, often seen in spring and autumn, should raise the possibility of B19 infection in the differential diagnosis of diffuse rashes and arthralgia in adults with HIV infection.

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